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OF MALARIA: A hypothesis
advanced by Richard Pfeifer
in 1892

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THE MODE OF TRANSMISSION OF MALARIA:

A HYPOTHESIS ADVANCED BY RICHARD PFEIFFER
IN 1892.¹

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OF the various conjectures on the mode of transmission of malaria which were made prior to the work of Manson and Ross, and the actual demonstration of the mosquito cycle of the parasite of this disease, that of Richard Pfeiffer appears to be the one most logically argued, most accurately deduced, and nearest the truth in the light of subsequent discoveries. Pfeiffer's remarks appear to have been entirely overlooked, probably because they were included in a paper dealing with another subject. The accuracy of Pfeiffer's reasoning is remarkable. It was based on the knowledge gained from a study of the common coccidium of the rabbit, and the scientific mind of Pfeiffer realised the possibility of the parasite of malaria having a similar life-cycle which completed itself, not on the ground in a protective oöcyst, but in the body of some blood-sucking invertebrate which might inoculate the germs thus produced into human beings. The similarity of the reproductive phase of the coccidia in the epithelial cells which they destroyed and that of the malarial parasite in the red blood corpuscles, together with the absence, in the case of the malaria parasites, of any resistant stage which could compare with the oöcysts of coccidia, led Pfeiffer to the view that in some blood-sucking insect lay hidden the secret of the transmission of malaria.

During the years 1889-90 this observer occupied himself with a study of the common coccidium of the rabbit. His paper, illustrated by excellent microphotographs, was published in 1892, and forms a landmark in our knowledge of these parasites. Hitherto it had been assumed that the sporulation of coccidia in the epithelium, and that in the oöcyst after it had left the body, represented two distinct parasites. To Richard Pfeiffer is due the credit of having first demonstrated that these were merely phases of one and the same organism. Sporulation in the epithelium led to multiplication within the host, while that in the oöcyst had to do with the spread of infection to another animal. The resistant oöcyst protected the

¹ Pfeiffer, R. (1892): Beiträge zur Protozoen-Forschung, I. Heft Die Coccidien-Krankheit der Kaninchen, Berlin.

parasite during its development outside the body. To Pfeiffer the endogenous sporulation of coccidia and malarial parasites were alike, while the absence of an oöcyst in the latter suggested the possibility of the exogenous cycle taking place while protected by some insect's body.

The following is a fairly literal translation of the final paragraph of Pfeiffer's most interesting and important work on the coccidia of the rabbit :—

As a result of the discoveries of Laveran, Marchiafava, Celli, and Golgi, it is known that in malarial fever the red blood corpuscles harbour characteristic motile amoeboid bodies which increase in size, bring about destruction of the occupied cell, and then, at regular intervals, as a result of segmentation associated with the formation of a residual body, produce numerous young individuals, which in their turn always give rise to a new generation of blood parasites. So far all is clear. But how does the parasite gain entrance to the human body? It is possible to produce the fever in healthy men, as was first shown by Gerhardt, by the injection of small quantities of blood from a person suffering from malaria. Without going into this matter further, it is clear that under no condition can this be the normal mode of infection. On the other hand, all epidemiological experience indicates a close connexion between marsh-fever and special conditions of the soil. The first and most evident idea was that in some way the blood parasite concerned was present in the soil or in the water of the infected locality, and that it attacked man as a result of inhalation or by way of the digestive tract. But all investigations which have been conducted along this line have remained without result. This is no longer surprising since we have learnt that the blood parasitising protozoa are extraordinarily delicate, and are entirely unsuited for a free life in the outer world. Grassi's hypothesis that the widely distributed amoebæ of marsh water enter the blood of man, transform themselves into malarial parasites and, as a result of adaptation to the existing conditions, lose their power of existing as free living organisms, is based on so forced a supposition and receives so little support from experiment, that it does not indicate a way out of the prevailing difficulty. How then is the undoubted dependence of malarial infection on environment to be explained? The following solution suggests itself, but I bring it forward only as a hypothesis, the justification for which rests in the fact that it indicates a line of investigation. It is possible that in the case of the malarial parasite also there exists an exogenous phase, a developmental cycle which completes itself outside the human host, possibly in the body of a lower animal (as, for instance, certain insects) and possibly also, in part at least, in the soil. This exogenous malarial germ could then be conveyed to man through the air or water, or as Robert Koch has remarked to me, through the sting of blood-sucking insects.

The reasoning is so sound, and has been so completely justified by subsequent investigations, not only on the life-history of the malarial parasite itself, but also by the discovery of the close relationship which exists between malarial parasites and coccidia, that it seems worth while to call attention to Pfeiffer's remarks.

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